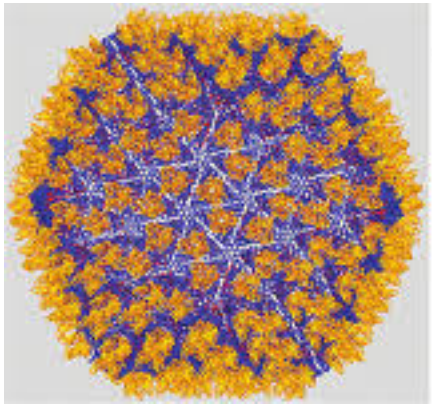
BIRNAVIRIDAE



Faculty
Dr. Rakhi Gangil
Department of Veterinary Microbiology
College of Veterinary Science and AH MHOW

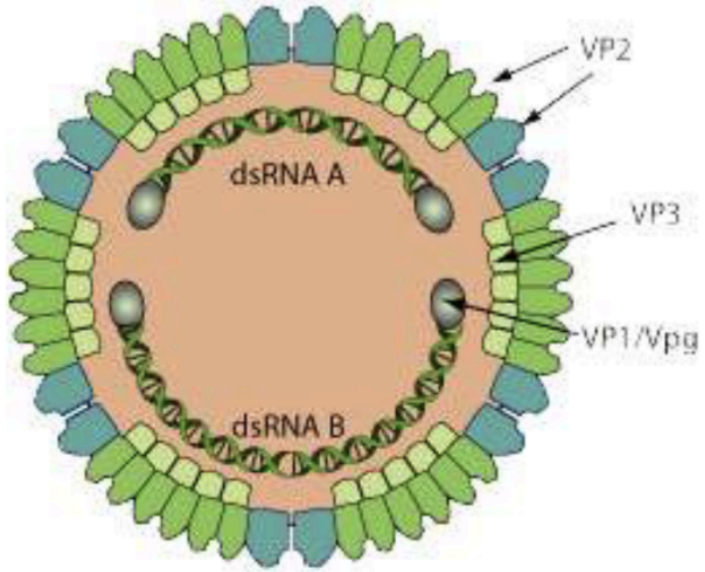
Classification

Family: *Birnaviridae*

- Avibirnavirus – Disease in birds, Infectious Bursal Disease
- Aquabirnavirus – Disease in aquatic animals, Infectious pancreatic necrosis
- Entomobirnavirus – Diseases in insects

Viral Characteristics

- Members of family *birnaviridae* are small and pleomorphic
- Non enveloped
- Icosahedral symmetry
- Double standard RNA (dsRNA)
- Size approx. 60 nm
- Genome of virus having two segments of linear dsRNA/ bisegmented



Continue…

- Over all genome size 6 kbp (Segment A larger-3.2 kb and Segment B smaller- 2.8 kbp in size)
  - Segment A synthesize 4 proteins – VP2, VP3 and VP4, VP5
  - Segment B synthesize 1 protein – VP1

VP2 protein is immunodominant protein and neutralizing antibody forms against it

VP2 protein used for serological diagnosis as well as formulation of new vaccine

VP1 (Polymerase)

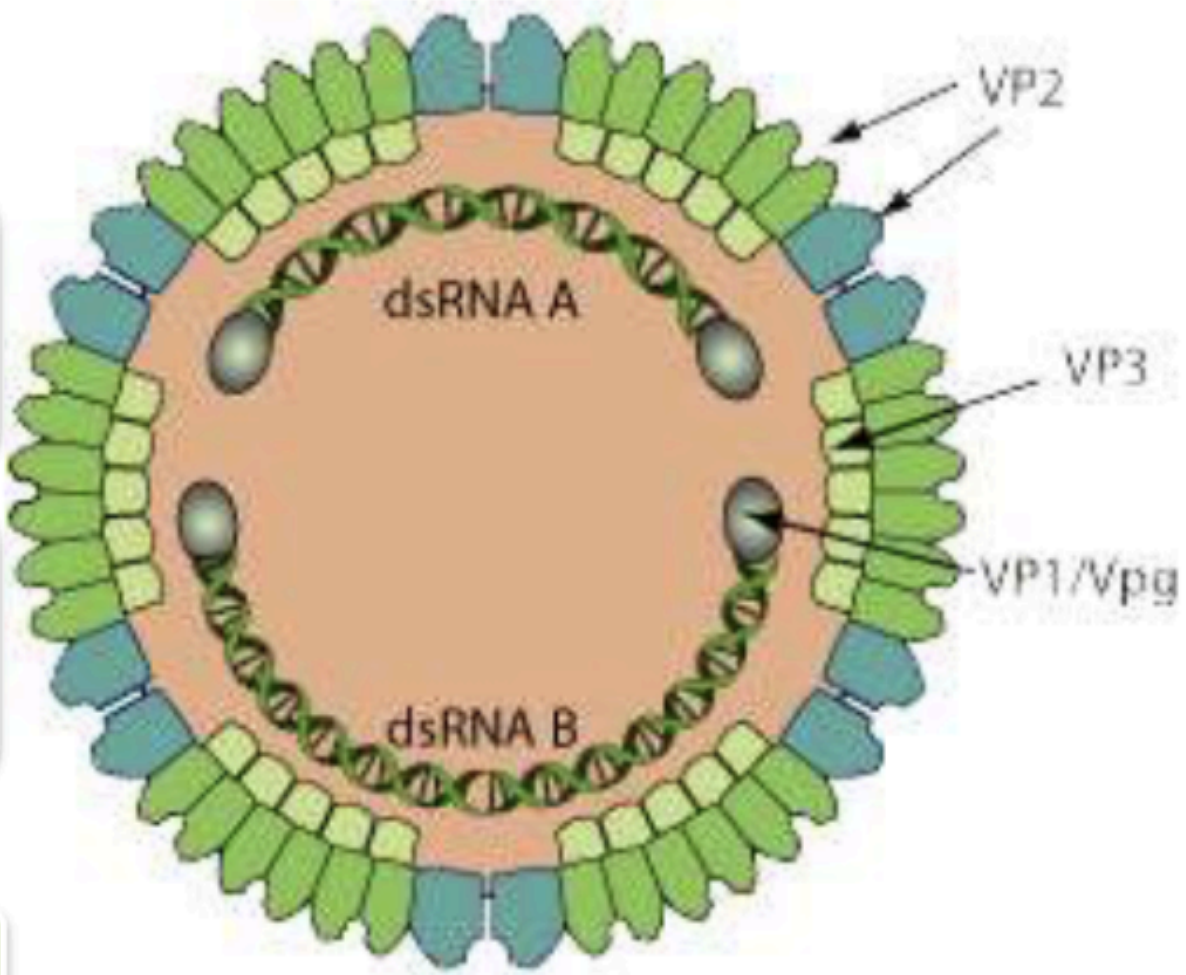
1. Encapsidation of the virus particle.
2. Speed of virus replication.

VP2 (external capsid)

encodes the major antigenic determinants of the virus, including epitopes that are important in virus neutralization.

VP 3 (internal capsid)

Interacts with both VP1 and VP2 and form VP1-VP3 complexes which is likely to be an important step in the morphogenesis of IBDV particles.



VP4 -VP 5

VP4

Is a minor and non-structural polypeptide.

VP5

likely has a regulatory function and plays a role in B-lymphocyte lysis.

Protein	Role
VP1 RNA dependant RNA polymerase Small amount in virus capsids	Encapsidation of viral particle
VP2 Main capside protein	Contain antigenic region responsible for : ➢ Serotype specific ➢ Elicit neutralizing antibodies
VP3 Other major structural protein not exposed at the surface	Morphogenesis of the virus
VP4	Viral protease (maturation of VP2 trimming peptides during virus assumbly
VP5	B-lymphocyte lysis.

Virus stability

The virus is very stable and survives for:

- 122 days in non disinfected house without birds.
- 52 days in contaminated water or feed.
- 5 hours at 56°C or 60°Cfor 1 hr
- The virus resists pH range from 3 to 9

Virus Replication

- Birnaviruses replicate in cytoplasm
- Virus penetrate into the cytoplasm
- Transcription of the dS RNA genome by virion associated RNA dependent RNA polymerase, occurs inside the virion, so that ds RNA is never exposed to the cytoplasm.
- This plus-strand transcript is used as template for translation
- (+) RNAs are ecapsidated in virion particle, inside which they are transcribed to give RNA (-) molecules with which they become base paired to produce ds RNA genomes.
- RNA replication is initiated independently at the ends of the both segments and proceed by strand displacement, with the inverted terminal repeats at the ends of each segment.

Infectious Bursal Disease (IBD)/ Gumboro disease

- The disease was first time reported in chickens at a village called Gumboro Delaware (USA) in 1957. So that it was named as Gumboro disease .
- The virus was first isolated in 1962.
- The disease is considerable economic importance because it replicate in the pre B lymphocyte in the bursa of fabricius leading to acquired B lymphocyte deficiency in affected birds.
- Disease is distributed all over the world.

IBDV Serotypes

- IBDV strains are classified into two distinct serotypes
- Serotype 1 – More virulent and produce more severe disease
  - ✓ Classical seroype 1 IBDV strain
  - ✓ Varient serotype 1 IBDV strain
- Classical serotype 1 and Varient serotype 1 do not cross protect each other
- Serotype 2 - mild and apathodenic IBDV strain, rarely produce disease

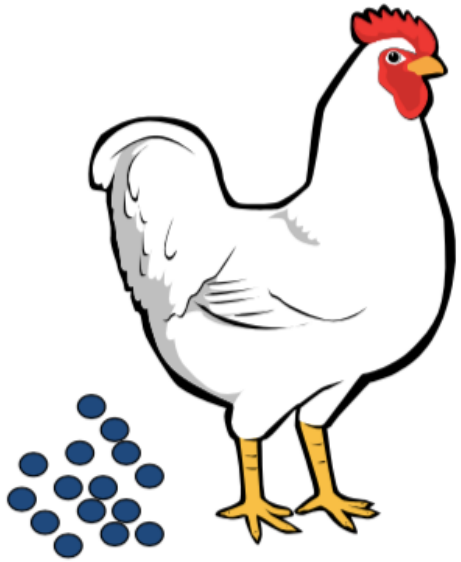
Epidemology

Host

- Chickens are primary host but ducks and turkey are also susceptible
- Birds Between age group of 3-6 weeks are more susceptible. Because upto 3 weeks of age maternal antibodies are present
- After 6 weeks bursa start regressing and virus cannot replicate

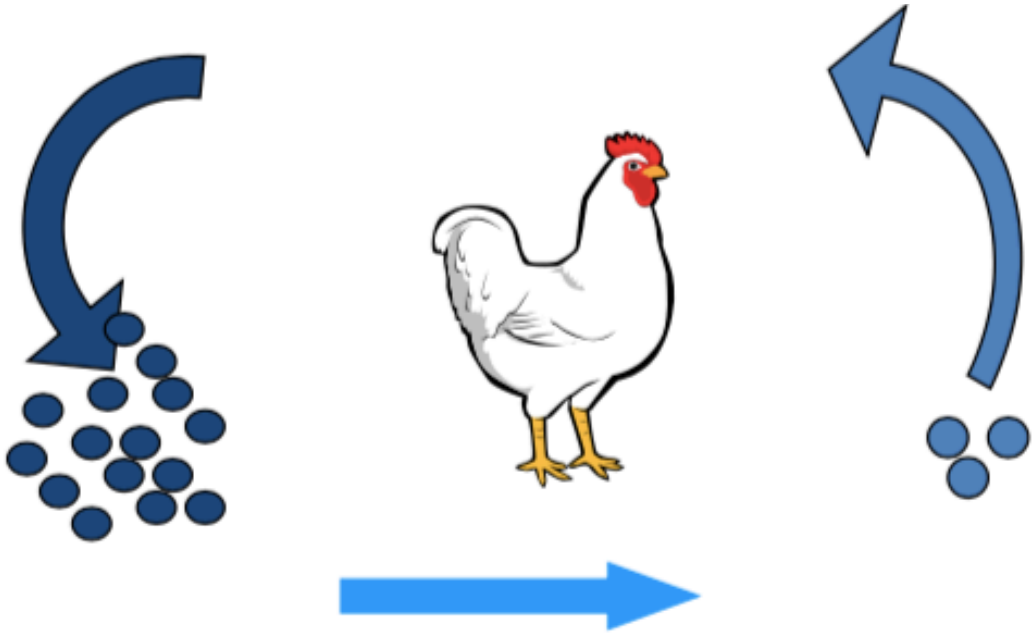
Transmission

- IBDV horizontally transmitted but not vertically.
- Infected and vaccinated birds usually shed virus through faeces from day 2 to till day 10 post infection or vaccination
- Horizontal transmission occurs through:
  - Oro faecal route
  - Contaminated equipment
  - Contaminated feed, water, dust, litter
  - Other organic material
- Mechanical transmission occur through insect vector (beetle - Lesser mealworm (*Alphitobius diaperinus*)



Continue..

Excretion
Day 2-10
post infection



Oral infection

Persistence
52 days in contaminated water or feed.

Pathogenesis of IBDV

Feco - Oral route infection

Virus enters to duodenum, ileum, jejunum and replicate in macrophages and lymphoid cells

↓

Primary viraemia through portal circulation

↓

Virus reaches to liver and replicated in Kupffer cells

↓

Virus spreads to bursa of fabricius 11 hours of post infection

↓

Active replication of virus in Bursal follicle and B cells

↓

Cont….

Leads to viral infection in organs like muscles, kidney and causing pathognomic clinical signs and death

Spreads to blood stream and causes secondory viraemia

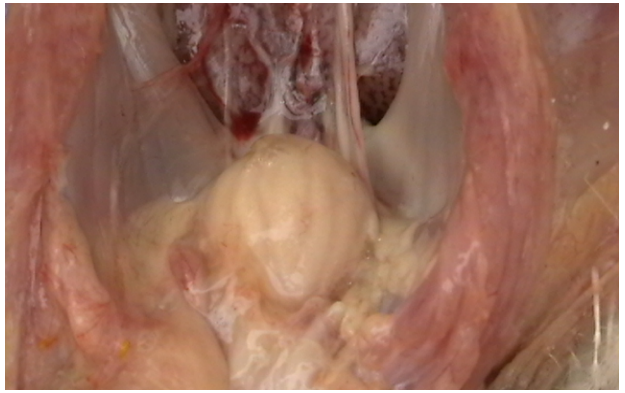
Selectively destroys B-Lymphocytes in Bursa

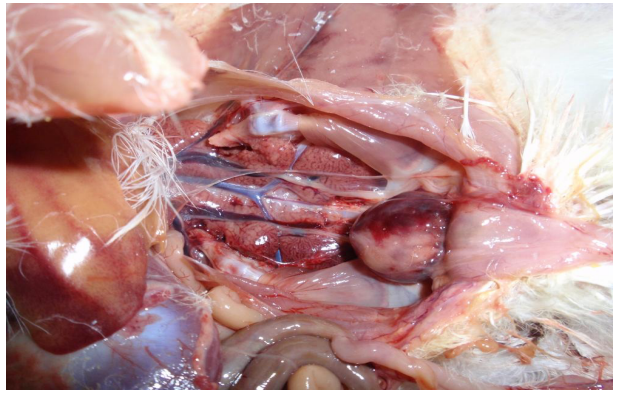
Leads to Immunosupression

(Main function of B lymphocyte to produce antibody. Large no of B cells destroys no antibody production)

Clinical Symptoms

- Fever, anorexia, dullness, depression, diarrhea, ruffled feather and death
- Morbidity is 100% and mortality sometimes reaches upto 90%
- Size of bursa enlarged 5 times to the normal and becomes edematous reddened or yellow or creamish coloured.





Diagnosis

- IBDV can be diagnosed by a combination of characteristic signs and post-mortem lesions
- Virus can be demonstrated by immuno-fluorescent technique in bursa of fabricius at about 4 days after infection
- Virus can be isolated in embryonated egg through Chorio-allantoic membrane route
- Isolated virus can be confirmed by Virus neutralization
- Antigen-capture enzyme- linked immunosorbent assays (ELISAs)
- AGID
- RT PCR

Prevention and control

- Strict hygiene and sanitization of the farms
- Two types of vaccines are available and cover both classical and variant strain
  - Live attenuated vaccine
  - Killed vaccine Killed vaccine can be given to chicks at 5-7 days of age
- After that live vaccine to be given at 3 week of age
- Then live vaccine is given before laying (before 18 weeks of age)